ETS EXPOSURE AND MONITORING

[43] **"Toxicology of Environmental Tobacco Smoke," M.J. Reasor. In: *Toxicology of Combustion Products.* L. Manzo and D.F. Weetman (eds.). Pavia, Fondazione Clinica del Lavoro, 71-76, 1992 [See Appendix A]**

This brief review discusses the chemistry of ETS and the use of biological markers to estimate exposure. The author also calls for research using animal models and in vitro tests to address the "controversy" surrounding the toxicology of ETS.

INDOOR AIR QUALITY

[44] **"Priority Among Air Pollution Factors For Preventing Chronic Obstructive Pulmonary Disease in Shanghai," X. Tao, C.J. Hong, S. Yu, B. Chen, H. Zhu, and M. Yang, *The Science of the Total Environment* 127: 57-67, 1992 [See Appendix A]**

In this study, Chinese researchers report on the comparison of sulfur dioxide levels, particulate levels, and indoor coal use with data on chronic obstructive pulmonary disease and lung function in residents of Shanghai. They conclude that use of coal indoors was the most important of the factors studied.

[45] **"Emissions of Volatile Organic Compounds from New Carpets Measured in a Large-Scale Environmental Chamber," A.T. Hodgson, J.D. Wooley, and J.M. Daisey, *Journal of the Air and Waste Management Association* 43: 316-324, 1993 [See Appendix A]**

In this study, VOC emissions from four new carpets were studied in an environmental chamber. Formaldehyde and 4-phenylcyclohexene (source of the "new carpet" odor) were identified, at levels which the researchers characterized as "low." The authors call for additional research on the potential of health effects from exposure to these VOCs.

SMOKING POLICIES AND RELATED ISSUES

[46] **"Clean Indoor Air Legislation, Taxation, and Smoking Behaviour in the United States: An Ecological Analysis," S.L. Emont, W.S. Choi, T.E. Novotny, and G.A. Giovino, *Tobacco Control* 2: 13-17, 1992 [See Appendix A]**

The authors of this study, two of whom are with the U.S. Office on Smoking and Health, compare state indoor air laws (smoking regulations) and data on cigarette consumption, smoking prevalence, and proportion of "quitters." They conclude that more restrictive regulations are associated with lower smoking prevalence and a higher proportion of quitters. They suggest that indoor air regulations could be used to "reduce tobacco consumption globally."

IN EUROPE & AROUND THE WORLD

REGULATORY AND LEGISLATIVE MATTERS

AUSTRALIA

[47] **Health Authority Considers Restaurant Smoking Ban**

The Eastern Metropolitan Regional Health Authority has reportedly recommended that the Health Commission impose a smoking ban in all South Australian restaurants, fast-food chains, cafeterias and hotels. The Health Minister apparently favors an approach to the issue that emphasizes education rather than legislation, and the Hotel and Hospitality Industry Association strongly supports self-regulation. The Government, however, has evidently warned the public that smoking may be outlawed in food outlets unless businesses provide smoke-free zones. *See Advertiser*, May 4, 1993.

[48] **Cab Drivers Respond to Taxi Smoking Ban**

Adelaide cab drivers are reportedly unhappy that the industry was not consulted before a ban on smoking in

South Australia's taxis and hired cars was adopted. However, according to the SA Taxi Association president, drivers and the industry generally favor a ban because of the "terrible risk" of health-related damages claims. The ban will apparently go into effect before the end of the year. *See Advertiser*, April 30, 1993.

AUSTRIA

[49] Shop Owners Protest Government Plans to Restrict Smoking

Some 10,000 news agents and tobacconists reportedly staged a one-day strike, closing their shops to protest Health Minister Michael Ausserwinkler's plans to impose smoking restrictions in public places. *See* issue 42 of this Report, March 5, 1993. According to a press report, one thousand demonstrators marched through the streets of Vienna, claiming that the measures could result in the loss of 30,000 jobs. *See The European*, April 29, 1993.

IRELAND

[50] Dublin Corporation Considers Workplace Smoking Policy

According to a press report, Dublin Corporation is assessing its workplace smoking policy in light of the settlement reached in the *Veronica Bland* case, wherein an employee received $21,500 for injuries allegedly caused by ETS exposure. *See* issue 40 of this Report, February 5, 1993. According to a spokeswoman, no similar claims have been filed against the corporation, which is currently conducting a staff survey to ascertain attitudes toward workplace smoking. A survey of this nature was undertaken in 1991, and, at that time, 93 percent of those surveyed favored restrictions on smoking in the workplace. *See The Irish Times*, April 16, 1993.

ETS/IAQ LITIGATION NOT INVOLVING CIGARETTE MANUFACTURERS

AUSTRALIA

[51] *Mansfield v. The Herald & Weekly Times Ltd.* (Victoria County Court, Melbourne) (filed November 19, 1992)

On April 29, 1993, the defendant's application to set aside the plaintiff's interlocutory judgment in default was heard and granted in this case, in which an employee claims workplace exposure to ETS caused his throat cancer. *See* issue 37 of this Report, December 18, 1992. The Master instructed the parties to agree upon a timetable for the filing of a defense.

LEGAL ISSUES AND DEVELOPMENTS

SINGAPORE

[52] Ministry of Health Conducts Workplace Smoking Seminar

According to a press report, the Singapore Ministry of Health conducted a seminar for senior management representatives of private companies about workplace smoking. An attorney speaking at the seminar reportedly advised participants that employers can legally impose rules against smoking in the workplace and cannot be sued by smokers if they do so. He also said employers cannot be sued by nonsmokers if no smoking restrictions are adopted. *See The Straits Times*, May 7, 1993.

OTHER DEVELOPMENTS

AUSTRALIA

[53] Author Addresses Scare Tactics Intended to Modify Behavior

According to a press report, retired physicist and senior federal bureaucrat Dr. John Farrands decided to tackle the issue of the use and abuse of science to create fear. In his book entitled "Don't Panic, PANIC!"

Farrands exposes what he considers to be improper attempts to modify behavior by "thought police" who base their efforts on fragmentary information. He apparently explains that such manipulation covers a whole range of issues, including smoking, diet and cholesterol. *See The Australian*, May 3, 1993.

[54] Doctors Disagree with Studies on Asthma and Pollution

According to a press report, doctors insist that colds and flu, housedust mites, pollens, molds and ETS are the primary triggers for asthmatic attacks. While studies overseas link ambient air pollutants to asthma, Australian doctors say the air quality in Australian cities has only a minor effect on the incidence and severity of asthmatic attacks. *See Sydney Morning Herald*, May 5, 1993.

[55] Survey Reveals 77 Percent of Top Companies Have Smoking Policies

The results of a national survey on workplace smoking policies were apparently recently published in 1992 in the booklet, "Workplace Policies and Programmes for Tobacco, Alcohol and other Drugs in Australia." The survey reportedly indicated that 77 percent of Australia's top 455 companies have introduced workplace smoking policies. Of the companies surveyed, some 46 percent impose a total smoking ban, 31 percent limit smoking to designated areas, and 23 percent had no policy. *See Financial Review*, April 29, 1993.

CANADA

[56] Canadian Carpet Industry Adopts Voluntary Testing Program

According to a news report, Canadian carpet manufacturers are adopting a voluntary carpet testing program already used in the United States. The program, soon to be implemented, calls for placement of a green tag to mark carpets that have been tested by an independent laboratory and found to emit less than 0.6 milligrams per square meter of volatile organic compounds per hour. However, Canadian government and consumer officials, consumer groups and those in the industry have been reported to say there have been very few complaints in Canada about carpets and negative health effects. According to a spokesman in Health and Welfare Canada's environmental health directorate, no clear link between carpet and serious health problems has yet been established. *See The Ottawa Citizen*, May 1, 1993.

HONG KONG

[57] Survey Reveals Support for Smoking Bans

According to a survey conducted by Hong Kong University for the Hong Kong Council on Smoking and Health (COSH), the majority of those surveyed favored bans on smoking in child care facilities, schools, workplaces and public places. Seventy-nine percent of those surveyed reportedly wanted nonsmoking areas to be designated in restaurants. Survey results have apparently been forwarded to the Health and Welfare branch, and the director of COSH hopes antismoking legislation will be introduced sometime this year. Some 1,222 people over the age of 18 were reportedly interviewed for the survey; approximately 12 percent were smokers. *See South China Morning Post*, April 28, 1993.

SINGAPORE

[58] Survey Finds Sick Buildings

According to a news report, SGS Singapore found five percent of 30 buildings tested were "sick." An organization dealing in inspection and control services, SGS Singapore reportedly conducted IAQ testing in 30 buildings, including offices, a shopping complex and two private homes. *See The Straits Times*, April 24, 1993.

UNITED KINGDOM

[59] BAT Publishes Brochure on "Social Engineering"

BAT has published a brochure entitled "Smoking: 'Fear of living' and Social Engineering in the Late Twentieth Century." The brochure focuses on what is perceived as the American preoccupation with risks and discusses the ways in which U.S. laws and litigation appear to be taking the enjoyment out of life. BAT concludes, "When the air outside cannot be breathed without choking in many cities around the world, when typhoid and cholera epidemics still rage in

places without adequate basic sanitation, it is sheer madness to become preoccupied with follies such as 'scent rape' and the hypothetical risks presented by a glass of wine at dinner or a whiff of someone's cigarette."

World Health Organization (WHO)

[60] WHO Meets In Geneva for 46th World Health Assembly

A debate on tobacco issues took place during the 46th World Health Assembly held in Geneva on May 3-14, 1993. Delegates from several countries reportedly observed that legislation, awareness campaigns and research studies were ongoing as part of general anti-tobacco activities. Forty-two countries apparently tabled a resolution calling on the WHO director-general to approach the United Nations secretary-general for purposes of urging him to ban the use of tobacco in buildings owned, operated or controlled by all UN organizations and specialized agencies. The resolution calls for progressive implementation of this ban within two years.

MEDIA COVERAGE

Canada

[61] "Tobacco Smoking in Aircraft — A Fog of Legal Rhetoric?," R.I.R. Abeyratne, *Air & Space Law*, No. 2 1993

The author of this article traces the efforts that have been taken in recent years by segments of the international aviation community to recognize the alleged adverse health and safety effects of smoking on airplanes. An extensive quote from an article by W. Allan Crawford about a 1988 study is set forth, including its conclusion that available scientific evidence does not support a ban on smoking in commercial aircraft.

The article acknowledges that "an aircraft in flight is a pressurized, airborne, air-conditioned, densely populated tourist and business facility at a high altitude with a relative humidity similar to that of Antarctica. Inside the aircraft, humans release on occasion hostile viruses and bacteria, shed dead skin particles, fungal spores and emit body odours." Yet, the author concludes that the "only known pollutant" in the aircraft is ETS.

Several cases from the United States involving nonsmokers' claims against the airlines are discussed, and the author suggests that the act of exhalation of tobacco smoke in an enclosed area could be classified as a tort and that regulators should take this into consideration when addressing the alleged problem of ETS in aircraft.

APPENDIX A

LUNG CANCER

[32] **"The Etiology of Lung Cancer," D.G. Davila and D.E. Williams, *Mayo Clinic Proceedings* 68: 170-182, 1993**

"A more complete understanding of the causes of lung cancer has long been an elusive goal of clinicians and basic scientists....[I]n contrast to many other malignant diseases, most cases of lung cancer can be prevented though avoidance of incriminated environmental factors."

"This review of the causes of lung cancer is the first contribution in a series of articles that will address the various clinical aspects of this disease. Herein we review some of the chemical and physical exposures, dietary factors, and lung diseases that have been associated with lung cancer. In addition, we discuss some of the recent studies that suggest a heritable predisposition to the disease and describe some of the cellular and molecular defects in lung tumors that are prominent factors in two proposed models of lung carcinogenesis. Finally, we comment on future directions of research in this area."

"Chemicals in tobacco smoke are the best-known lung carcinogens; this association has been noted for more than 50 years."

"Although the incidence of lung cancer among those who passively inhale environmental tobacco smoke is clearly increased, the exact level of increased risk is unknown and controversial...'Passive smokers' or nonsmokers who breathe environmental tobacco smoke are thought to inhale qualitatively similar tobacco smoke contents but quantitatively lesser amounts."

"Assigning a definitive level of increased risks for lung cancer from passive exposure to tobacco smoke has been problematic because no truly unexposed control group exists for comparison. Hence, differences in risk between those known to be exposed to environmental tobacco smoke and the so-called control groups are likely to be small and difficult to demonstrate."

"Overall, odds ratios for lung cancer among passive smokers have been estimated to be 1.24 (95% confidence interval, 1.04 to 1.5) in case-control studies and 1.44 (95% confidence interval, 1.2 to 1.72) in prospective studies."

"Despite the limitations of some of these studies, the 1986 Surgeon General's report, the National Academy of Sciences, and the International Agency for Research on Cancer all formally recognize the increased risk of lung cancer from passive exposure to smoke...The most recent review from the Environmental Protection Agency of the health effects of passive smoking as it pertains to lung cancer is in preliminary draft form and will likely influence policies about smoking in the workplace."

"The causes of lung cancer will continue to be studied at the epidemiologic level. Population-based prospective studies will focus on determining potential dose-response relationships between lung cancer and environmental tobacco smoke with use of better measures of exposure."

[33] **"Implications for Disease Misclassification in Epidemiological Studies of Lung Cancer Risk for Nonsmokers Exposed to Environmental Tobacco Smoke," A.W. Katzenstein, *Environment International* 19: 211-212, 1993**

"The U.S. Environmental Protection Agency (EPA) appears to have diminished or dismissed the potentially significant role of disease misclassification in its review of lung cancer risk in nonsmokers reportedly exposed to environmental tobacco smoke (ETS)...EPA made no attempt to adjust for potential disease misclassification, which is more likely to result in overestimation than underestimation of relative risk."

"Disease misclassification arises when 'lung cancer cases' are not primary lung carcinomas but are secondary cancers that have metastasized to the lung from primary tumors originating in other body tissues. Definitive diagnosis of primary lung cancer requires histological or cytological examination of lung tissue."

"The potential effect of disease misclassification can be illustrated in the study of Hirayma (1984) in Japan, which was among the earliest of the epidemiological assessments said to find lung cancer risk in nonsmokers married to smokers."

"EPA reported a crude relative risk of 1.38 (90% C.I. 1.03-1.87). There is potential disease misclassification, however, for all 200 lung cancer cases, with more than four and one-half times as many cases among those exposed to ETS than among those not exposed."

"If as few as four of the 163 lung cancer cases exposed to ETS were disease misclassified — i.e., were not primary lung carcinomas — the relative risk would be a statistically nonsignificant 1.34 (C.I. 0.99-1.71)."

"Of the 31 epidemiological studies in the data base of the EPA Review Draft, all lung cancer cases of 12 studies were reported to have been histologically or cytologically confirmed. In 14 studies, from 2% to 100% of the lung cancer cases had not been definitively confirmed, while in 5 studies, the method and extent of verification were not reported."

"For all case-control studies where the extent of diagnosis confirmation was established, EPA data show 302 lung cancer cases not definitively confirmed."

"The EPA Review Draft shows 64% of the cases were classified as exposed to ETS, so that there may be nearly twice the likelihood of disease misclassification among 'cases' classified as exposed to ETS as among those not exposed. However, any assumptions about the distribution of disease misclassifications in specific studies or in the overall data base would be entirely speculative and insupportable."

"The failure to give appropriate consideration in the EPA Review Draft to the significant potential for disease misclassification reduces confidence in risk estimates derived from the data and raises serious questions about public health policies based on such studies."

Cardiovascular Issues

[35] **"Carbon Monoxide and Cardiovascular Disease: An Analysis of the Weight of Evidence," J.H. Mennear, *Regulatory Toxicology and Pharmacology* 17: 77-84, 1993**

"The mechanism(s) [through] which either active or passive smoking might increase risk of cardiovascular disease have yet to be unequivocally defined. A prominent and frequently mentioned cause or contributor is the production of myocardial ischemia through exposure to ETS-associated carbon monoxide. The purpose of this review is to weigh the evidence relative to the hypothesis that ETS-related exposures to carbon monoxide (CO) can contribute to either the initiation or exacerbation of ischemic cardiovascular disease in humans. The results of this review show that there is little clinical or experimental evidence that is relevant to the issue and that that which is available does not support a role for ETS-associated carbon monoxide in the causation or exacerbation of ischemic heart disease in non/never-smoking humans."

"Carbon monoxide, produced during the incomplete combustion of all organic materials, is the most extensively studied and best understood component of either mainstream or sidestream cigarette smoke. This gas avidly competes with oxygen for binding to hemoglobin (Hb). The combination of CO with HB results in the formation of carboxyhemoglobin (COHB) and compromises the transport of oxygen to the tissues of the body."

"Overall, the results of studies in humans afford some evidence that exposure to extremely high concentrations of CO may elevate risk of ischemic heart disease and decrease the exercise tolerance of people with coronary artery disease. Such effects are consistent with the production of systemic anoxia and impaired myocardial oxygenation. However, it remains to be established whether ETS can contribute sufficient environmental CO to impact on the cardiovascular status of either healthy or compromised humans."

"It has been frequently and correctly noted that sidestream tobacco smoke contains a higher concentration of CO than does mainstream smoke...American cigarettes are recognized to deliver approximately 15 mg/cigarette of CO via mainstream smoke and 50 mg/cigarette via sidestream smoke."

"This relatively high concentration in sidestream smoke has led many to conclude that ETS is a major contributor to environmental CO concentrations. Such a conclusion is not supported by the results generated in field studies during which the air in residences, work places, and public places has been analyzed under both smoking and nonsmoking conditions."

"On the basis of the available data obtained from field studies, it is clear that ETS contributes CO to the environment. However, the increment of environmental CO attributable to tobacco smoking is exceedingly small. Further, this small increase is easily masked by normal day-to-day variations in ambient concentrations which are attributable to the presence of other CO sources such as automobiles and the combustion of heating and cooking fuels."

"While conducting this analysis no attempt was made to directly address the issue of whether or not exposure to ETS per se causes or exacerbates cardiovascular disease. The results of this review have established, however, that if the purported impact of ETS on cardiovascular disease is real, it can be neither explained nor mediated thorough ETS-associated increases in ambient concentrations of carbon monoxide. There is scant evidence to support a role for carbon monoxide in the causation of ischemic heart disease. Further, the results of field studies of air quality in nonsmoking and smoking homes, offices, and public places demonstrate that ETS contributes only minor and toxicologically insignificant increments in ambient carbon monoxide concentrations. These increments are variable and easily masked by other commonly encountered carbon monoxide sources such as internal combustion engines and the burning of cooking and heating fuels."

Respiratory Diseases and Conditions — Children

[36] **"Child Day Care, Smoking by Caregivers, and Lower Respiratory Tract Illness in the First 3 Years of Life," C.J. Holberg, A.L. Wright, F.D. Martinez, W.J. Morgan, L.M. Taussig, and Group Health Medical Associates, *Pediatrics* 91(5): 885-892, 1993**

"Day-care attendance has been associated with an increased risk of hospitalization for lower respiratory tract illnesses (LRIs). This study examines, in a health maintenance organization population of children, the associations between child day care and the occurrence of LRIs in the first 3 years of life. Smoking by caregivers and a possible protective effect of longer day-care enrollment in relation to LRIs are also addressed."

"Information on day-care arrangements was elicited from 1006 parents of infants for five age intervals in the first 3 years of life: birth through 3 months, 4 to 6 months, 6 to 12 months, 1 to 2 years, and 2 to 3 years. Data on LRIs in the first 3 years of life were recorded by pediatricians at the time of the acute illnesses."

"This study has shown that the risk of LRI increases up to twofold or more for children, between 4 months and 3 years of age, who are in child care situations involving the presence of three or more unrelated children. The association is independent of other implicated risk factors, including type of and time spent in the child care setting, maternal education, number of others sharing the child's bedroom, having other siblings, parent history of respiratory trouble, maternal smoking, smoking in the child care setting, gender, and ethnicity. Beyond the threshold of three or more unrelated children in the child care setting, there is no evidence in this population to suggest that increasing numbers are associated with increased LRI risk. Prior to 4 months of age, an increased risk associated with the number of others present is not apparent. At this younger age, the risk is associated simply with being in any child care setting other than at home."

"After controlling for other risk factors, the presence of siblings was associated with risks of LRI of similar magnitude to those of exposure to unrelated children in the child care setting, but only in the first 6 months of life. This would imply that although the nature of the risk associated with unrelated children or other siblings is similar, the source of the contacts is associated with a different risk profile. Thus, our results suggest that while there is a constant ongoing risk associated with exposure to unrelated children, in the first 3 years of life, the LRI risk associated with having other siblings is present only during the first year, and then it decreases, possibly becoming protective in the second and third years of life. This suggests there is a more limited exposure to infection associated with the presence of the same group of children, since the total number of contacts is reduced."

"After controlling for other risk factors, including maternal smoking, the present study has further demonstrated an increased risk for wheezing LRIs of up to threefold or more in the third year of life in those infants who are in a child care setting with a smoking caregiver. To our knowledge, this is the first demonstration of a passive smoking effect in children attributable to sources outside of the home environment. The reason why this effect would be seen in the third year of life is not apparently due to an increase in the amount of time spent in the care setting, as we had initially thought. However, the majority of infants in their third year of life in another home setting with a caregiver who smoked were also with a caregiver who smoked in their second year of life. This suggest that

prolonged exposure to environmental tobacco smoke may increase the risk of wheezing LRIs."

"Over the first 3 years of life approximately one fifth of infants of nonsmoking mothers were in a care setting with a smoking caregiver. Also, we find that infants of mothers who smoke are more likely to be placed in a child care setting with a smoking caregiver compared with infants of nonsmoking mothers. There were relatively few heavily smoking mothers who placed their child in a care setting where there was no smoking."

"Given the continued widespread utilization of child day-care, the findings of this study suggest that the risk of LRIs would be reduced in care settings involving fewer than three unrelated children. This could be particularly relevant in the first 6 months of life, when the incidence of LRI is highest. In addition, child day-care in the absence of environmental tobacco smoke would decrease the risk of LRIs."

[37] **"Risk Factors for Developing Wheezing and Asthma in Childhood,"** W.J. Morgan and F.D. Martinez, *Pediatric Clinics of North America* 39(6): 1185-1203, 1992

"Wheezing respiratory illness and asthma are responsible for a significant proportion of both acute and chronic illness in childhood. Affecting approximately 5% to 10% of children, asthma is of growing concern because of an apparent increase in mortality and morbidity. The risk factors associated with the development of wheezing illness and asthma have therefore been the focus of much investigation in the last two decades."

"This article first reviews risk factors for wheezing in infancy and the toddler years. The possibility that early viral-related wheezing illness predisposes to later asthma and chronic lung dysfunction is then discussed. With this background, risk factors for the development of persistent asthma in later childhood are explored, with particular attention to the role of allergy in the pathogenesis of chronic airway inflammation and asthma."

"Both exogenous factors deriving from the child's life experience and endogenous (congenital) factors may increase the risk of wheezing in infancy."

"Parental, particularly maternal, cigarette smoking has been clearly associated with an increased risk of wheezing, respiratory symptoms, lower respiratory tract illness, and hospitalization in exposed infants....The effect of maternal smoking has been assumed to be due to passive inhalation of sidestream tobacco smoke by the infant. This might then result in airway inflammation and other alterations favoring both viral infection and the development of clinical wheezing illness or pneumonia. Recently, preliminary results from several studies have suggested that this relationship also may be due to alteration of the developing lung by maternal smoking, leading to a greater risk for wheeze with infection. Maternal smoking during pregnancy results in fetal stress secondary to both intrauterine hypoxia and nicotine exposure. Whereas the growth retardation associated with maternal smoking during pregnancy is well known, lung-specific effects may occur as well, including a reduction in lung elastin content."

"[A] significant proportion of children (25%) wheeze before 3 years of age, but only a minority go on to develop asthma. This remission in wheezing illness may occur in some children because of lung growth and development. In other children, however, the presence of allergy and other factors leads to the development of asthma independent of prior wheezing lower respiratory tract illness history. Risk factors for the development of asthma in later childhood are discussed here."

"[T]here is convincing evidence that an allergic inflammatory reaction occurring in the airways is almost certainly necessary for the development of asthma in children more than 5 years of age. Moreover, the severity of asthmatic symptoms may be directly related to the degree of sensitization to aeroallergens. A better understanding of the mechanisms by which allergy causes asthma may help to identify predisposing factors for asthma."

"Several studies have suggested that children exposed to environmental tobacco smoke in their homes may be at increased risk of developing asthma....Not all studies have been able to confirm these findings, but the number of cigarettes smoked and the educational level of the mother were not always adequately controlled in the negative studies. It is thus quite likely that exposure to cigarette smoke may cause asthma in susceptible children."

"The mechanisms by which environmental tobacco smoke may cause asthma are not well understood. Recent studies in Italian schoolchildren showed

increased sensitization to aeroallergens, increased IgE levels, and increased prevalence of eosinophilia in children of smoking parents. Environmental tobacco smoke also may enhance bronchial responsiveness, and this effect is apparently independent of that of passive smoking on allergic sensitization."

"Wheezing lower respiratory tract illness in infancy and asthma share the clinical findings of wheezing and respiratory distress. Although the link between wheezing lower respiratory tract illness in infancy and the subsequent development of asthma is a limited one, both conditions do share some common risk factors, including exposure to environmental tobacco smoke, difficult living conditions (low socio-economic class, crowding, allergen exposure), and increased risk in males....Although the endogenous risks for these two outcomes may be fixed, it is clear that caregivers may help to reduce or eliminate the exogenous risks listed earlier by parental education and improvement of the living conditions of young children."

OTHER CANCER

[38] **"Risk Factors for Renal Cell Carcinoma: Results of a Population-Based Case-Control Study," N. Kreiger, L.D. Marrett, L. Dodds, S. Hilditch, and G.A. Darlington,** ***Cancer Causes and Control*** **4: 101-110, 1993**

"It has only been in the last few years that renal cell carcinoma has become the focus of epidemiologic research, and many questions about risk factors remain. The study reported here was designed to elucidate the importance of a number of potential risk factors, including active and passive cigarette smoking, body mass, diet, use of analgesic and diuretic medications, and hormonal effects."

"This population-based case-control study encompassed the province of Ontario, and included all newly diagnosed, histologically confirmed, cases of renal cell carcinoma diagnosed in 1986 or 1987, who were aged 25-69 years and resided in Ontario at the time of diagnosis. Cases were ascertained through review of pathology reports received by the Ontario Cancer Registry."

"Passive smoking was not confined to cigarette smoke, but included exposure to cigar and pipe smoke as well. Subjects reported their usual passive exposure in three categories (<3, 3-8, or <8 hours per day) combining home and workplace exposures."

"The risk associated with passive smoking was assessed among never-smokers only. In males, no statistically significant effect was observed; in females, inclusion of passive smoking resulted in a statistically significant improvement in the model. The highest exposure level (more than eight hours' passive smoking per day) was associated with a risk of 1.6 (CI = 0.5-4.7) for males, and 1.7 (CI = 0.8-3.4) for females."

"Our data show that for female smokers, in contrast to male smokers, the reported inhalation of tobacco smoke is an important variable. Consistent with this sex difference is the difference in the effects of passive smoking: in females, passive smoking significantly increased risk; while in males, there is no significant effect of passive smoking. This finding is in concordance with a report of passive smoking and cancer risk in adults showing greater relative risk among groups with lower overall cancer risk. It is possible that the effects of inhaling and of passive smoking may be more difficult to detect in men, given the smaller numbers of male nonsmokers with high levels of passive smoke exposure. Women who smoke do so at lesser amounts than men, and may inhale smoke differently, leading to a greater relative contribution to total exposure of passive smoke or to a greater potential for passive smoke to have an effect on risk."

"In summary, we have confirmed the association between active cigarette smoking and increased risk of renal cell carcinoma among both males and females and have provided data suggesting passive cigarette smoking to be of importance. Our data indicate that 41 percent of renal cell carcinoma in males, and 28 percent in females, may be attributed to ever having smoked cigarettes. In addition, while dietary intake, particularly of fats, does not account for a large percentage of renal cell carcinoma in Ontario, possibly 17 percent in males and 26 percent in females may be attributed to having a high [body mass index] at some time in life. Given these attributable risks, there is considerable potential for reduction of the incidence of renal cell carcinoma through modification of these factors."

[39] **"Parental Smoking and Risk of Childhood Brain Tumors," E.B. Gold, A. Leviton, R. Lopez, F.H. Giles, E.T. Hedley-Whyte, L.N. Kolonel, J.L. Lyon, G.M. Swanson, N.S. Weiss, D. West, C. Aschenbrener, and D.F. Austin, *American Journal of Epidemiology* 137(6): 620-628, 1993**

"We used data obtained in one of the largest, population-based case-control studies of childhood brain tumors, to undertake the present set of analyses, to assess the role of parental smoking in the risk of the most frequently occurring solid tumors in children. The size of the study and the level of detail of parental smoking information obtained permitted an in-depth investigation of this important question with a high level of statistical power."

"Cases were identified from eight population-based Surveillance, Epidemiology, and End Results (SEER) program tumor registries, representing a combined annual catchment population of 4.72 million children."

"Information on demographic characteristics, occupational history, personal and family medical histories, and habits including smoking was obtained from each parent for both cases and controls in a structured interview in the home. Interviews were completed for 361 children with brain tumors and 1,083 control children."

"No significant differences were found between cases and controls in maternal or paternal smoking at any time or specifically during the year the index child was born (including both the prenatal and early postnatal periods) or 2 years before the index child was born, which also included the preconception period. These analyses were stratified by parental educational level to control for its potential confounding effect, which was found to be nil. The potential confounding effects of alcohol, coffee, and tea consumption were also examined and not found to affect in any substantial way the lack of observed effect of parental smoking on risk of childhood brain tumors. The estimated relative risk associated with smoking more than one pack per day also was not significantly greater than that for smoking less than one pack per day."

"Maternal smoking, or exposure to cigarette smoke during pregnancy, has been associated with brain tumors in children in one study, but not in others. The negative studies are similar to the present study in that they: 1) interviewed and incorporated information about fathers, 2) identified controls randomly from the population, and 3) individually matched controls to cases. The positive study is the largest study prior to the present one but interviewed only mothers of cases and of friend and neighborhood controls."

"However, one possible explanation for the discrepant findings is when the data were collected. The one study that found an association between childhood brain tumors and maternal exposure to sidestream smoke was conducted years before the studies that did not find this association. Our data were collected during the same time that the null studies were conducted."

"If the time period that the data were collected accounts for the observed difference, then at least two inferences are possible. One is that with the recent decline in the prevalence of maternal smoking has come a loss of statistical power. Another inference is that the number of pregnant women who smoke has not really declined, but only the truthful acknowledgement of cigarette smoking has declined."

"[O]dds ratios for all brain tumors, as well as for astrocytoma and medulloblastoma, tended to be close to unity, and thus provide no evidence for any increased risk associated with maternal or paternal smoking prior to or during pregnancy or with passive exposure to parental smoking postnatally. While these findings obviously do not outweigh the many other significant health hazards to parent and child associated with parental smoking, the consistency of these findings in one of the largest studies of childhood brain tumors provides strong evidence that parental preconception or pre- or postnatal smoking does not affect the risk of brain tumors in the offspring."

Other Health Issues

[40] **"Effects of Maternal Smoking Upon Neuropsychological Development in Early Childhood: Importance of Taking Account of Social and Environmental Factors," P.A. Baghurst, S.L. Tong, A. Woodward, and A.J. McMichael, *Paediatric and Perinatal Epidemiology* 6: 403-415, 1992**

"Since the possible long-term effects of maternal smoking on childhood neuropsychological development are of great theoretical and practical importance,

the question has emerged of whether the reported lower neuropsychological functioning of children can be attributed to exposure to maternal smoking or to other coexistent aspects of the child's social and environmental circumstances."

"The results reported in this paper come from a follow-up study of 548 4-year-old children whose mothers were recruited during pregnancy....The analyses focus on the associations of the neuropsychological outcome in childhood with maternal smoking, and a number of key social and environmental factors. Evidence is provided that the decrements in children's neuropsychological functioning associated with antenatal or postnatal exposure to maternal smoking are not as large as those attributable to the child's social and environmental factors, and that appropriate adjustment for these factors may explain the mild association between exposure to maternal smoking and neuropsychological development in children."

"The analyses of children's scores on the...scales of children's abilities with postnatal exposure to maternal smoking showed that the children of smokers performed at 2.4 to 4.1% lower level in most of the testing sessions by comparison with those of nonsmokers....The decrements in...scores in the children of smokers were statistically significant. The children with postnatal exposure to maternal smoking also had lower scores for...verbal, perceptual-performance and motor subscales."

"However, no significant differences were found between the scores of children with antenatal exposure to maternal smoking and those of children with no antenatal exposure."

"These results reveal a statistically significant inverse association between maternal smoking and neuropsychological development which becomes quite insignificant when other putative determinants of development are taken into account. Interpretation of these findings is therefore difficult, and must take into account both the reliability of the exposure measures, and the more general problems of selection bias, and 'over-adjustment'."

"[S]ocio-economic status, the home environment provided by the parents and maternal IQ all make significant inroads into the crude association of poor development with maternal smoking, and corroborates the results of earlier studies. While it may still be argued that smoking reduces a woman's IQ and renders her less able to provide a highly stimulatory home environment for her children, there is little published evidence on which to base such speculation."

"We conclude that there is, at present, no strong evidence that maternal smoking exerts an independent effect upon neuropsychological development in early childhood. A major reason for the inconsistent results observed in this area may be confounding due to social and environmental factors. If passive smoking does have an effect on the development of children's abilities, it is likely to be difficult to detect in the presence of wide variations in these factors. In order to gain a clearer understanding of this problem, more precise measures of exposure to environmental tobacco smoke, both *in utero* and postnatally, may also be required."

[41] "Smoking and the Sudden Infant Death Syndrome," E.A. Mitchell, R.P.K. Ford, A.W. Stewart, B.J. Taylor, D.M.O. Becroft. J.M.D. Thompson, R. Scragg, I.B. Hassall, D.M.J. Barry, E.M. Allen, and A.P. Roberts, *Pediatrics* 92(5): 893-896, 1993

"One way to assess the importance of passive smoking is to examine the effect of smoking by the father and other household members on the risk of SIDS. This paper reports the effects of maternal smoking during pregnancy and the effects of smoking by the mother, father, and other household members after the infant's birth."

"Infants of mothers who smoked during pregnancy had a fourfold greater risk of SIDS than infants of mothers who did not smoke. Infants of mothers who stopped smoking during pregnancy had a lower risk of SIDS, but this was not statistically significant. Infants of mothers who smoked in the previous 2 weeks had an increased risk of SIDS compared with infants of nonsmokers. Furthermore, the risk increased with increasing levels of maternal smoking. Similarly, infants of fathers who smoked in the previous 2 weeks had an increased risk of SIDS, but a dose effect was not evident. The number of smokers in the house (parents and other household member) also increased the risk of SIDs, as did the presence of smoking by other household member, excluding the parents."

"The relationship of maternal smoking status with other variables was examined in the control group. As expected, maternal smokers as a group were significantly more likely to be of lower socioeconomic status, Maori, of lower educational level, unmarried, younger at first pregnancy, younger at the birth of the infant, late attenders at antenatal classes, and nonattenders at antenatal education classes; to have infants of lower birth weight; not to breast-feed; and to share the bed with their infant. Maternal smokers did not differ from nonsmokers for infant's sex, number of previous pregnancies, gestation, admission to neonatal unit, season, and infant's sleeping position."

"After controlling for region, time of day, season, marital status of mother, socio-economic status, ethnic group of infant, mother's age at birth of infant, infant's sex, birth weight, age of infant, breast-feeding, sleep position, and infant sharing bed with another person, we found that maternal smoking was still significantly associated with an increased risk of SIDS (OR = 1.65; 95% CI = 1.20, 2.28), as was smoking by the father (OR = 1.37; 95% CI = 1.02, 1.84)."

"The number of smokers in the household was associated with a significantly increased risk of SIDS after control for potential confounders (1 household smoker OR = 1.12, 95% CI = 0.77, 1.63; 2 smokers OR = 1.75, 95% CI = 1.23, 2.48; 3+ smokers OR = 2.07, 95% CI = 1.26, 3.41). The presence of smoking by others in the house (excluding parents) was not a significant risk factor after smoking by the mother and father and other potential confounders were controlled."

"The effect of smoking by the father increased the risk of SIDS if the mother smoked, but not if she did not smoke."

"Maternal smoking increased the risk to the baby substantially, but lack of breastfeeding had a further and independent effect."

"The effect of smoking by the father has not been examined in detail previously. Although one study has shown smoking by the father to be a risk factor for SIDS, that study did not control for maternal smoking. This is essential as we have shown that maternal and paternal smoking behaviors are related. We found that the increased risk of SIDS from paternal smoking persisted after controlling for maternal smoking and their potential confounders. We were unable to demonstrate a dose-response curve for paternal smoking. This may have occurred because much of the father's smoking is done away from the house. But when the combined effects of parental smoking are considered, it appears that the father's smoking increases the risk of SIDS when the mother smokes, but not if she does not smoke. We did not expect the lack of effect of father's smoking when the mother is a nonsmoker, but it may be that a nonsmoking mother is more likely to insist that a smoking father smoke away from the infant."

"Finally, we address the central question: 'Is smoking causally related to SIDS?' Criteria for causation in an observational study such as this are as follows:"

"• Temporal relationship where the putative risk factor precedes the event. This criterion is obviously fulfilled, particularly since information on smoking during pregnancy was collected in obstetric records prior to the death."

"• Consistency of the findings. Maternal smoking has been identified as a risk factor for SIDS in many studies."

"• Strength of association. The stronger the association, the more likely the risk factor is causally related. In this study different measures of maternal smoking have ORs greater than 4, which is moderately strong."

"• Biological gradient. In this study a biological gradient was seen for the amount the mother smoked, the number of smokers in the household, and possibly, the duration of smoking in pregnancy."

"• Biological plausibility. Smoking during pregnancy reduced birth weight, a risk factor for SIDS. Furthermore, maternal smoking in pregnancy may contribute to chronic fetal hypoxia, which may predispose to SIDS. Passive smoking in the infant's first year of life increases the risk of respiratory infections. Infections may result in pyrexia and lead to hyperthermia if the infant sleeps prone or is excessively dressed. An alternative hypothesis is that maternal smoking may damage the fetal brainstem, resulting in an abnormal respiratory response to noxious stimuli, hence increasing the risk of SIDS. This has some support from an animal model."

"Thus all the major criteria for causation are met."

[42] **"Smoking, Passive Smoking and Smell," P. Hepper, *Medical Science Research* 20: 265-266, 1992**

"In recent years there has been much interest in the effects of passive smoking, or environmental tobacco smoke, on health. As yet, however, there have been no investigations of the effects of passive smoking on smell. This experiment examined the effects of smoking and passive smoking on olfactory performance."

"Subjects were divided into three groups. Smokers were individuals who smoked between 20 and 30 cigarettes per day, and had been smoking for at least 6 months. Nonsmokers were individuals who had never smoked and were kept out of a smoky environment for at least 10 h prior to the test. Passive smokers were individuals who had never smoked but for 1 h preceding the experiment had sat in a room with other smokers (the rest room of the university library)."

"Subjects were taken into a well ventilated room and presented with odours in increasing strengths . . . Subjects were asked to sniff deeply and inform the experimenter of what they smelt. The experimenter recorded the strength at which the subject correctly identified the odour."

"45 subjects (15 per group) were tested using peppermint and 30 (10 per group) using lemon. Smokers were tested 5 min after finishing their last cigarette and passive smokers 5 min after leaving smoky environment."

"Smoking exerted a highly significant effect on olfactory performance....[S]mokers required a stronger concentration to identify the odours than both passive smokers and non-smokers. Passive smokers required a stronger concentration than nonsmokers."

"To examine the 'permanence' of the deficit caused by smoking, two further experiments were carried out using peppermint as the stimulus....For the first group, smokers and passive smokers were tested 1 h after finishing their last cigarette or 1 h after leaving the smoky atmosphere, respectively. In the second group, smokers and passive smokers were tested 24 h after finishing their last cigarette or leaving the smoky atmosphere, respectively."

"There was a highly significant effect of smoking. As with the previous experiment, smokers were poorer at identifying the odour than passive smokers, who were in turn worse than non-smokers."

"[One hour] refraining from smoking or being out of a smoky environment had little effect on olfactory performance. However, for passive smokers 24 h out of a smoky environment returned their olfactory performance to normal. No recovery of performance was observed for smokers."

"The mechanism of smoke-induced deficits in olfactory performance has yet to be elucidated. It is possible in these studies that different mechanisms operated in groups of smokers and passive smokers. The reduction in performance of passive smokers may have been due to a short-term habituation effect, whereas the effects in smokers resulted from more permanent alterations in the olfactory mucosa or receptor cells, which would correspondingly take longer to reverse."

ETS Exposure and Monitoring

[43] **"Toxicology of Environmental Tobacco Smoke," M.J. Reasor. In: *Toxicology of Combustion Products.* L. Manzo and D.F. Weetman (eds.). Pavia, Fondazione Clinica del Lavoro, 71-76, 1992**

"Environmental tobacco smoke (ETS) is a complex and dynamic mixture of particles and gases which has been poorly characterized. Most experimental research has involved study of sidestream smoke rather than ETS, therefore, such results are difficult to interpret relative to human exposure."

"Biological markers, including cotinine in biological fluids and DNA and protein adducts, have been utilized to assess exposure to ETS; however, none has been identified that can serve as a quantitative surrogate for ETS. As a result of the paucity of information regarding ambient ETS characterization and exposure assessment, it has been difficult to evaluate the possible toxicological effects of ETS on humans."

"Toxicological aspects concerning ETS exposure in humans are an area of ongoing debate and controversy. A number of reports have appeared alleging that chronic exposure to ETS results in adverse health effects in children and adults. A body of literature exists which has provided strong scientific reasoning in dispute of that conclusion. The principal reason for this controversy involves the nature of the human studies which have been almost exclusively by epidemiological procedures. Epidemiology is notoriously

weak at establishing causal relations at the low relative risks reported in studies involving ETS exposure."

"It is unlikely that this controversy will be resolved by dependence on further epidemiological studies; alternative approaches will have to be utilized including studies using animals and 'in vitro' systems. In contrast to the abundance of epidemiological studies concerning ETS exposure, virtually no relevant information exists on the effects of ETS in animals and 'in vitro' systems. In studies using animals, the protocols generally have involved exposure to only sidestream smoke and at levels that are unrealistically high compared to ambient exposure to ETS. As a result, it is difficult to interpret the results of these studies in the context of human exposure. Increased emphasis in ETS research should be placed on developing and utilizing whole animal and 'in vitro' exposure systems and protocols utilizing conditions simulating ambient exposures."

"It has been suggested that ETS is just a dilute form of the mainstream smoke inhaled by the active smoker, and therefore, in attempting to understand the possible effects of ETS, it is valid to extrapolate from what is known about active smoking. There is no evidence to support such an assertion. While mainstream smoke is highly concentrated, and its properties are rather well characterized, ETS is exceedingly more dilute and far more dynamic. Thus it seems apparent that comparison of ETS exposure to active smoking in a toxicological context is of little value."

Indoor Air Quality

[44] **"Priority Among Air Pollution Factors for Preventing Chronic Obstructive Pulmonary Disease in Shanghai," X. Tao, C.J. Hong, S. Yu, B. Chen, H. Zhu, and M. Yang, *The Science of the Total Environment* 127: 57-67, 1992**

"Chronic obstructive pulmonary diseases including chronic bronchitis, asthma and emphysema, are some of the major causes of death in residents of Shanghai city proper where ambient air pollution is mainly from sulphur dioxide (SO_2) and inhalable particulates and indoor air pollution is mainly from the use of coal for heating and/or cooking. The problems that city environmental protection planers face are how important these exposures are in relation to COPD in local residents and which factor should be controlled urgently. The purpose of our study is to determine the control priority among ambient SO_2, IP and indoor use of coal to prevent COPD in residents of the city."

"Distribution of ambient SO_2, and IP concentrations were described using a...imulation. When stratified by two extreme levels of ambient SO_2 and IP and types of fuel used indoors, eight local area populations in four communities with different combinations of exposure levels were selected. In each community a local area population mostly using coal and one mostly burning gas was chosen. Chronic obstructive pulmonary diseases including chronic bronchitis, asthma and emphysema, are a major cause of death in residents of Shanghai. The relationship between the three air pollution factors and their health effects were analyzed at the level of mortality, prevalence of symptoms of COPD, lung function and non-specific immunologic function."

"Our study suggests that the indoor use of coal is more important than ambient SO_2 and IP in relation to mortality and prevalence of COPD, pulmonary symptom, lung function and non-specific immunologic function in residents of Shanghai city. We recommend that the change of fuel from coal to gas or other types which produce less pollutants should be given priority over the effort to reduce ambient SO_2 and IP, and this should be conducted first in the high ambient SO_2 and/or IP areas."

Indoor Air Quality

[45] **"Emissions of Volatile Organic Compounds from New Carpets Measured in a Large-Scale Environmental Chamber," A.T. Hodgson, J.D. Wooley, and J.M. Daisey, *Journal of the Air and Waste Management Association* 43: 316-324, 1993**

"Since little was known about the quantitative emissions of VOCs from carpets, this study was undertaken to measure chamber concentrations, emission rates and mass emissions of individual VOCs released by new carpets that are typical of the major types of carpets used in residences, school classrooms and offices. Four carpets, including two with SBR [styrene-butadiene rubber] latex adhesive and two with other types of backings, were selected for study. Concentrations, emission rates and mass emissions of VOCs from samples of these carpets were measured under simulated indoor conditions in a 20 m^3 environ-

mental chamber over a period of one week following the installation of a sample in the chamber. Duplicate chamber experiments were conducted for one carpet. Concentrations of selected compounds emitted by samples of the carpets in 4 L chambers were compared to the corresponding large chamber results to evaluate the usefulness of much smaller chambers. In addition, the concentrations and emission rates of VOCs emitted by a new carpet installed in a house were measured over a period of seven weeks."

"The emission of VOCs from the study carpets were [sic] low relative to many other types of indoor sources, such as architectural finishes....Within this context of relatively low emissions, those compounds that had the highest emission rates or mass emissions were evaluated for their potential to produce health and comfort effects."

"The eight dominant compounds identified...are styrene, 4-PCH [4-phenylcyclohexene], formaldehyde, vinyl acetate, 2,2,4-trimethylpentane, 1,2-propanediol, 2-ethyl-1-hexanol, and BHT [butylated hydroxytoluene]. Of these, the most is known about the toxicity and irritancy of formaldehyde. Formaldehyde is a strong sensory irritant."

"[S]ince other sources of formaldehyde are often present in buildings, the addition of a carpet source could result in concentrations that approach or exceed lower limits for irritancy. Only very limited data are available on the toxicity and irritancy of the other compounds at low concentrations."

"Odor is an important factor that influences people's acceptance of products used indoors. The 4-PCH in SBR carpets is the source of the 'new carpet' odor which some people find objectionable. This odor, by itself, may be a source of complaints by consumers."

"It is not clear whether the emissions of VOCs from carpets could produce health or comfort problems like those reported by some consumers. On the one hand, the emissions of TVOC from carpets are low relative to other sources of TVOC that are commonly found in buildings. On the other hand, the potencies of different VOCs may vary over a number of orders of magnitude as evidence by the ranges of Threshold Limit Values for industrial exposures to chemicals, sensory irritancy as measured by the mouse bioassay and odor thresholds. New SBR carpets are likely to produce an odor for a period of several months due to the persistent emissions of 4-PCH. With the exception of formaldehyde, only very limited data are available on the irritancy and toxicity of the compounds emitted by carpets. However, it is possible that several of the dominant compounds, in additional to formaldehyde, are sensory, and possible respiratory, irritants at relatively low concentrations. Therefore, it would be of value to determine the sensory and respiratory irritancy of these compounds, as well as their neurotoxicity, using appropriately sensitive tests."

SMOKING POLICIES AND RELATED ISSUES

[46] **"Clean Indoor Air Legislation, Taxation, and Smoking Behaviour in the United States: An Ecological Analysis," S.L. Emont, W.S. Choi, T.E. Novotny, and G.A. Giovino, *Tobacco Control* 2: 13-17, 1992**

"[S]tatewide smoking restrictions and increases in cigarette excise taxes represent two potentially powerful public health tools that may influence smoking behaviour. This investigation examined the association of state clean indoor air laws and state excise taxes on cigarettes with these measures of smoking behaviour: current smoking prevalence, proportion of quitters, and consumption of cigarettes per head."

"All 50 states and the District of Columbia were categorized according to the scope of their clean indoor air law in 1989. Such laws ranged from nominal policies, in which smoking was regulated in three or fewer public places, to extensive policies, in which smoking was regulated in four or more public places plus restaurants and private workplaces."

"[S]moking prevalence was inversely related to the degree of restriction of the clean indoor air policy. The average smoking prevalence was 28% in states without clean indoor air laws and 24% in states with extensive clean indoor air laws. Average cigarette consumption per head, following a pattern similar to that of smoking prevalence, was about 119 packets in states without clean indoor air laws and 105 packets in states with extensive clean indoor air laws. The proportion of smokers who had stopped smoking (quitters) was positively associated with the scope of the clean indoor air policies. The average proportion of quitters was 44% in states without clean indoor air policies and 50% in states with extensive clean indoor air policies."

"Although we could not test the causal relation between clean indoor air legislation and measures of smoking behaviour in our study, our multivariate analyses indicated that either moderate or extensive clean indoor air laws (but not nominal or basic clean indoor air policies) were associated with a lower smoking prevalence and a higher proportion of quitters. Even though the primary purpose of implementing clean indoor air policies is to protect the non-smoker from exposure to environmental tobacco smoke, such policies — particularly comprehensive policies — may have an impact on smoking behaviour."

"The impact of clean indoor air laws and cigarette excise taxes on smoking behaviour deserves further evaluation. In addition to clean indoor air legislation and excise taxes, there are a number of other public health strategies that can be used to reduce tobacco consumption globally. These include regulating tobacco advertisements and promotions, using mass media in a coordinated anti-smoking campaign, and providing school health education programmes on smoking....Only through coordinated global tobacco control initiatives will we curtail what is surely expected to be a worldwide epidemic of smoking-related morbidity and mortality."